

Hepatic Encephalopathy because of Falciparum Malaria in a Child: A Rare Association

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Abstract: *Hepatic encephalopathy due to Plasmodium falciparum hepatitis rarely reported in adult patients and also a rare incidence in a child with mixed of Plasmodium malarial parasite. We report a case of hepatic encephalopathy in falciparum malaria in child . Prompt early diagnosis and initiation of antimalarial treatment can have reduce the mortality. This is the first case of Hepatic encephalopathy due to Plasmodium falciparum infection from Eastern India, in our opinion.*

Keywords: Hepatic Encephalopathy, Falciparum Malaria, child

1. Introduction

Hepatic dysfunction in malaria has been reported in literature , but hepatic encephalopathy is almost never seen in children with Falciparum malaria[1].Patients with hepatocellular dysfunction in malaria are more prone to develop complications, but have a favorable outcome if hepatic involvement is recognized early and managed properly [2].

2. Case Report

A 9 year old girl admitted with fever for five days, yellowish discoloration of body for last 3 days and drowsiness since last night. The girl was healthy previously. There was no history of convulsion or unconsciousness. The sleep wake cycle of the girl was altered. On examination, the girl was disoriented, febrile and icteric. There was also mental and motor slowing. Abdominal examination revealed no abnormality except tender hepatomegaly.

Investigation revealed: Hemoglobin 11.6gm%. Reticulocyte-1%. TLC-8600/cmm. Plasmodium falciparum is present in peripheral smear, P.falciparum antigen in blood was positive. Bilirubin-2.5mg/dl (Normal<1mg/dl), SGPT-199 U/L(Normal<40) , Alkaline Phosphatase-1351 U/L(Normal<644), PT-14 sec (INR-1.2). Serum electrolytes and creatinine was normal. Viral markers for hepatitis A, B, C and E were negative. G6PD estimation, Serum Ceruloplasmin and antinuclear antibody , anti-smooth muscle antibody , liver/kidney microsomal antibody -1 were normal .The girl was treated in a intensive care setting with i.v quinine and supportive management. With treatment the girl become fully conscious within 3 days, icterus decreased gradually and repeated investigation showed a gradual normalization of serum bilirubin and liver enzymes. The girl was discharged after 10 days in stable condition.

3. Discussion

In this case, clinically there was encephalopathy with hepatic involvement. Laboratory report confirms the same, with five times raised SGPT and more than two times raised alkaline phosphatase. There was no evidence of hemolysis. The case was also diagnosed as falciparum malaria. Other possible common causes of hepatic encephalopathy has been excluded.

In falciparum malaria jaundice can be explained by several mechanisms [3]. It can result from either severe hemolysis or hepatic involvement. Infestation of the red blood cells by P. falciparum and resultant hemolysis results in the rising bilirubin. The rupture of hepatocytes during the primary schizogony results in cellular damage. Sequestration of the parasite-infested red blood cells in the capillaries causes clogging of the capillaries in the important organs and the resultant ischemia can lead to organ system dysfunction. When the same happens in the liver capillaries, acute hepatic dysfunction ensues. In falciparum malaria parasites are clogged in the capillaries and it is often not possible to find it in peripheral smear. Severe coagulopathy is almost never seen in isolation with severe malaria and prothrombin time is usually within normal limits even in patients with marked elevation of liver enzymes [4].

Hepatic encephalopathy because of Plasmodium falciparum hepatitis reported from in adult [5] and a child from with mixed of Plasmodium vivax and Falciparum infection [6].

Finally , Plasmodium falciparum can be present with hepatic Encephalopathy and Quinine is the mainstay of treatment ; early treatment can reduce mortality.

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